ARTICLE V.

CALCULOUS AND OTHER AFFECTIONS OF THE PANCREATIC DUCTS.[1]

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THE earliest writer who mentions the subject of Pancreatic Calculi is Regnero de Graaf, who wrote in 1671. Since then instances have been met with from time to time, and recorded with more or less accuracy by writers both in Europe and in this country, the most recent being a case under the charge of Dr. William Pepper, at the Hospital of the University of Pennsylvania, in 1880. Altogether we have been able to collect *thirty-five cases* in which, upon post-mortem examination, stony concretions were found in the pancreas. We cannot but believe that calculi are present in the pancreas far oftener than is supposed, and we can only attribute the paucity of medical literature upon the subject to the inexperience or carelessness of the diagnostician or pathologist.

In spite of the many difficulties which always attend the efforts of a collaborator, we have succeeded, we think, in obtaining a fair amount of material upon this subject, and we have endeavoured in these "Extracts" to summarize as much as possible the results of our work upon the pathological anatomy and symptomatology of calculous and a few allied affections of the pancreatic ducts.

I. PATHOLOGY AND MORBID ANATOMY.

1. *Varieties of Calculous Concretions found within the Pancreas.*—Before proceeding to a minute description of the calculous formations having their origin within the pancreas, it is well at the outset to refer briefly to the varieties into which such formations can be classed, and to mention—

a. Free concretions,
b. Calculous incrustations of the duct walls, and
c. "Acne pancreatica,"

as three general heads, under one or the other of which, all the instances of pancreatic calculi, contained in the reported cases, can easily be grouped. By far the greater number of stony formations observed belong to the class of free concretions; but the other two conditions, though rare, have been sufficiently well described to render the distinctions between the classes clear.

a. Free Concretions.—It is difficult to give any generalization based upon the observed cases of free concretions, as the older writers adopted the most arbitrary standards of comparison, and as the reports of later authors are to a great extent incomplete. We have attempted to record, in the form of a table, all the instances which it has been possible to collect, in such manner as will most clearly present their many differences and peculiarities to the eye of the reader. (See table, pp. 406 and 407.)

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[1] Extracts from Inaugural Thesis, University of Pennsylvania, 1882.

b. Calculous Incrustations of Duct Walls.—It occasionally happens that the inorganic constituents of the pancreatic juice, instead of being precipitated in the form of a free concretion, crystallize upon the walls of the ducts.[1] Calculous incrustations are thus formed, which occur as single points, or plates, or else layers covering the whole duct wall.[2] The origin of the two hollow concretions, described by Henry and Matani, can possibly be explained in this way,[3] although that mentioned by the latter seems to be more of the nature of a free concretion, and has been considered as such. And yet both were large, adherent to the interior of the pancreas, were hollow, and contained within their cavities a fluid (in the one case milky-white, in the other green), in which small granular concretions were suspended.

It is well known that one of the changes which occur in the walls of the so-called *kyste confirmé* is a chalky precipitation, sometimes so extensive as to cover its whole internal surface.[4] In one of the cases reported by Recklinghausen, the cyst found in the pancreas was beyond question the result of the complete occlusion of the duct of Wirsung (by a calculus), and its subsequent dilatation. The contents of this cyst were made up in great part of altered pancreatic secretion, and its walls were studded in some places with thick white plates; in others with grayish layers, which glistened like mother-of-pearl; while in others still they were covered only by a thin film.[5] It seems to us that this description agrees exactly with that already given of lime incrustations upon the inner wall of the pancreatic ducts, and it does not appear unlikely that we have here an example of such an incrustation formed simultaneously with the production of the calculus, and before any dilatation of the duct

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[1] Klebs, Handbuch der Patholog. Anat., Berlin, 1876. Pankreassteine. 1 Bd., s. 544. ......Delafield, Handbook of Post-mortem Examinations and of Morbid Anatomy, New York, 1872: The Pancreatic Ducts, p. 203.

[2] Klebs, ibid.

[3] Matani, Naturen genees-kundige Bibliotheek—door Eduard Sandifort, Gravenhage, 1765: Waarneeming van Antony Matani, over eene steenagtige samengroejing van het alvleesch, in het lyk van een' mensche gevonden; or, Gottingische Anzeigen von Gelehrten sachen, No. 10, 1765. Antonii Matani observatio de lapidea pancreatis concretione in humano cadavere reperta.......Henry, Journal de chémie médicale, iv. série, Paris, 1855: Recherches analytiques sur une concrétion particulière du Pancréas, tome i. p. 273; or, France médicale et pharmaceutique, 3e Année, Paris, 1856: Sur les concrétions que présente le pancréas, No. 6, p. 42.......Klebs, ibid.

[4] Klebs, op. cit. s. 547.

[5] Recklinghausen, Virchow's Archiv, Berlin, 1864: Auserlerne pathologisch-anatomische Beobachtungen, I. Drei Fälle von Diabetes Mellitus, a. 30 Bd., s. 360.

Author.	Date.	No. of calculi.	Weight.	Size.	Shape and surface.	Colour.	Friability	Chemical analysis.	Remarks.
De Graaf,	1671	7 or 8	....	"Large as chick pea."					
Mercklin,[1]	1678	1	....	"Larger than an almond."	Irregular and sharp edges.	1 part black			
Eller,	1755	1	....	"Considerable."					
Galiati,	1758	20	....	"Little."					
Matani,	1765	1	ʒij	"Thick as little finger of full grown person."	Irregular, like a piece of coral.	White, yellow, coppery spots	......	Fused, became black, lost weight. Soluble in aqua fortis and solution of silver, not in alkalies; *yellow* precipitate with Hg_2Cl_2.	Odour of foul water; no taste.
Fournier,	1776	9	....	....	Triangular.				
Cawley,	1788	Pancreas full of calculi.	....	Varied; none larger than a pea.	Irregular, like mulberry stones	White			
Salmade, (Portal's 1st)	1797–8	12	....	Varied; some large as hazel-nut.	Round.	Whitish	......	Powder freely soluble in hot water.	Taste flat and insipid like saliva.
Baillie,	1812 1833	A number.	....	Kernel of hazel-nut.	Irregular.	White	......	Soluble in HCl; CO_2 extricated. Composed of carbonate of lime.	Pemberton, 1830, examined one of these, and found it composed entirely of carb. of lime.
Elliotson,	1833	Pancreatic and larger branch ducts crammed.	....	....	....	White			
Bigsby, Bonetus.	1835	4							
Schupman, 1	1841	1	ʒiij ℈j	Length 1–6 lines; diam. 5–6 lines.	Cylindrical, irregular, covered with project'ns.	Grayish or yellowish white			
" 2	....	2	℈j	Length a few lines.	Irregular.	White			
Elliotson,	1846	100 at least.	....	....	Similar to Baillie's drawings.				
Gould, 1	1847	In cyst wall several; in duct 3; tail filled.	....	Very minute; diam. 3–4 lines.	Rough.	....	......	Made up of carbonate of lime.	
" 2	....	Several.	....	Pea.					
Clayton,[2]	1849	Numerous; one escaped.	....	Length ⅔ inch.	....	....	......	Carbonate of lime, traces of phosphate of lime, animal matters, and fat.	
Dufrèsne, 1 Portal's 2d	1851	In cyst many pan. and biliary cal.	....	Granular.	Unequal.	....	Hard		

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[1] In Mercklin's case, three large and several small pancreatic calculi were found in an abscess in right lumbar region.

[2] In Clayton's case, one pancreatic calculus escaped into peritoneal cavity; it was two-thirds inch long, and consisted of carbonate of lime, traces of phosphate of lime, animal matter, and fat.

Author.	Date.	No. of calculi.	Weight	Size.	Shape and surface.	Colour.	Friability	Chemical analysis.	Remarks.
2 Muhlbauer	....	In pancreatic abscess great variety of calculous matter.	....	From grain of sand to size of lentil.	....				
Clark,	1851	Mass of calculous matter.	....	....	Irregular and rough.	Glistening white	......	Consisted probably of carbonate and phosphate of lime.	
Virchow,	1852	Twice met with concretions in excretory duct of pan.	....	....	....	....	Half soft	Consisted probably of hard insoluble protein, which had great similarity of reaction with that contained in semen. Identical with the kind of albumin described by Bernard.	
McCready,	1856	1	....	Length 1 inch; diam. ⅓ inch.	....	....	......	Composed of mixed carbonate and phosphate of lime.	
Recklinghausen, 1	1864	1	....	Bean.					
2(a)	....	Small particles of calculous matter suspended in whitish tenacious fluid.	....	....	....	....	......	Carbonate and phosphate of lime.	
(b)	....	2	....	(1) 1 inch long, ⅜ inch thick. (2) 1⅛ inch long, ⅜ inch thick. (3) Smaller particles.	Spindle-shaped. Grauu'r surface distinguished later (2).	Grayish white	Brittle		
Cornil and Ranvier,	1869	Not specified.	....	....	Irregular.	White	Friable	Carbonate and phosphate of lime.	The calculi were in a thick, white pulp, and probably wall incrusted.
Janeway,	1872	A number.	....	Small.	Irregular.	Whitish	Fragile	Probably of carbonate and phosphate of lime.	
Curnow,	1873	19. Ducts and smaller radicles studded.	....	Varied; length ⅓ to 1 in.; breadth $\frac{1}{5}$ inch.	Worm-eaten appearance.	Whitish, when rubbed had enamel lustre; glistening, porcelain-like fracture	......	Organic matter 24 parts. Fixed salts 76 parts. 100 parts. Chiefly carbonate of lime.	
Pepper, U.H. Reports	1880	Pancreas involved in a large tumour containing calculi.	....	....	....	....	......	....	Along course of duct were cysts, some containing calculi.

NOTE.—The description of pancreatic calculi embodied in this Table are as far as possible in the words of the authors quoted.

occurred. If this is so, many of the incrustations found upon the inner wall of pancreatic retention cysts are the result of the precipitation of the inorganic constituents of the secretion upon the duct walls prior to their dilatation, and therefore should not be ascribed to the later changes occurring in the cystic walls, nor be compared to the results of a prolonged endo-arteritis.[1]

c. *Acne Pancreatica.*—The condition, sometimes met with in the pancreas, described by the pathologist Klebs, and called by him Acne Pancreatica, seems to have almost entirely escaped the notice of former investigators,[2] and Klebs himself fails to mention it in his article upon pancreatic concretions.[3] The pathological process involved consisted in a change of the normal pancreatic secretion into a fatty, chalky pap.[4] In the only case observed with any care, the exterior of the gland was covered by large light-yellow spots, which, like the acini, occupied a certain depth, and consisted of a thick, smeary, butter-like substance containing fat. The pancreatic duct was dilated and filled, as were the accessory ducts, with a pap-like mass, which contained pus elements and light particles of hardened protein.[5] Yellow spots similar to those above referred to, and having the appearance of blisters, were found by Klebs upon the surface of an enlarged pancreas. It is believed that these were cysts formed by the dilatation of the smaller branch ducts, and the clear or clouded semifluid mass which they contained was found to be composed partly of fat-globules, and partly of some cretaceous material. These spots may easily be mistaken for abscesses; for those small cysts which are only occasionally present in the acini, and are of no pathological importance; and, finally, for the small spots (from which chalk or tufts of fat-crystals can be expressed) found in a gland which has been the seat of chronic pancreatitis from obstruction of the portal vein.

Only a few other cases have been met with which seem to us illustrative of the condition just described, viz., acne pancreatica. In them either the pancreatic ducts were found full of a chalky powder,[6] of an earthy, doughy substance,[7] or else the whole gland was converted into a tophaceous or calculous mass.[1] It may be here remarked that the chemical composition of such deposits differs in no way from that of free concretions.

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[1] Klebs, op. cit. s. 547.

[2] Klebs, op. cit. s. 547.......Gendrin, Histoire Anatomique des Inflammations, Paris, 1826: Pancréatites chroniques, tome ii. p. 263.

[3] Klebs, op. cit. s. 544.

[4] Gendrin, ibid.

[5] Virchow, Verhandlungen der med. physik. Gesellschaft zu Wurzburg, 1852: Zur pathologisch-anatomischen Casuistik, 4, III. Bd., s. 366, cf. II. Bd., s. 53.......Klebs, op. cit. s. 547.......Friedreich, Ziemssen's Encyclopedia of Medicine, New York, 1878: Diseases of the Pancreas, Cysts, vol. viii. p. 615.

[6] Wilson, Medico-Chirurgical Transactions, London, 1842: An account of a case of extensive disease of the pancreas, vol. xxv. p. 42.

[7] Schmitt, Zweifelhafte Schwanger, Wien, 1818.......Klebs, op. cit. s. 547.......Cornil et Ranvier, d'Histologie Pathologique, Paris, 1869–1873: Kystes, t. ii. p. 974.......Cornil and Ranvier, Manual of Pathological Histology, translated by Shakespeare and Simes, Phila. 1880: Cysts, p. 581.

The cause of the formation of chalky masses or concretions in the pancreatic ducts, or in the small retention cysts above described, is without doubt either a chemical alteration, brought about in the secretion of the gland through contact with the inflamed duct walls, or else an obstruction offered to the free outflow of the secretion by a local interstitial inflammatory thickening, or a catarrhal swelling of the lining membrane of the duct of Wirsung, or of any of the accessory ducts. The question of causation will, however, be dwelt upon at greater length in a later section.

2. *Location of Calculous Deposits.*—The seat of the various concretions found within the pancreas can, we believe, be most clearly shown by the subjoined analysis. It may be mentioned, however, that, in the only instance of calculous incrustation of the duct wall in which the seat of this incrustation is specified, the part of the duct of Wirsung situated midway in the gland was alone affected. With regard to the condition spoken of as acne pancreatica, a sufficiently detailed description has been already given. We will proceed then to the remaining class, viz., Free Concretions.

Free Concretions.

	Seat.	No. of instances.
I. Found in duct of Wirsung only.	Exit	3
	Middle	1
	Tail	...
	Not specified	3
	Total	7
II. Found in branch ducts and in duct of Wirsung	Exit	...
	Middle	1
	Tail	1
	Throughout	7
	Not specified	2
	Total	11
III. Seat not specified	Total	3
IV. Found in abscess in pancreas	Head	1
	Centre	1
	Total	2
V. Found in cyst of pancreas	Head	3
VI. Exceptional cases	In abscess in right lumbar region	1
	In peritoneal cavity	1

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[1] Meckel, Koreff, Dissertatio inaug. med. sist. theoret. consid. icteri. Halæ Magd., 1759, § 12, p. 16.

3. *Mode of Origin of Pancreatic Concretions.*—Although the origin of pancreatic concretions depends upon some special functional or structural alteration in or about the gland, yet we must consider these, in some cases, as the mere local manifestations of some general morbid condition. Disregarding the study of these general conditions, it will be necessary in the present section to refer to such local alterations only as are immediately connected with the production and development of these concretions. For the sake of clearness we will divide these alterations into two classes, namely: Changes in the gland itself, or in the chemical composition of its secretion; and Changes in the structure or relation of the surrounding tissues or organs.

Among the many modifications, both functional and structural, of which the pancreas is the seat, those only are directly connected with the formation of calculi which tend to prevent the free outflow of the pancreatic juice. A morbid change in any part of the gland structure,[1] especially at its head;[2] a mucus plug,[3] or an already formed calculus lodged in the duct of Wirsung or any of the larger ducts; a catarrhal[4] or chronic inflammatory condition with thickening of the same;[5] an interstitial inflammation with hyperplasia of connective tissue;[6] or a biliary calculus which has found its way into the pancreatic duct;[7] are some of the many causes which go far to explain the origin of pancreatic calculi. The conditions are varied, it is true; the effects the same. Let there be an obstruction, a compression, a complete occlusion of the duct internal to the gland, and the secretion is dammed back, it accumulates and stagnates when it should escape, and those conditions most favourable to the precipitation of its inorganic constituents are all thus furnished. But who can say that there are not other and more remote causes, in addition to those which have been already mentioned? Is compression or complete occlusion of the ducts necessary? In atrophy, in morbid softening, especially in fatty metamorphosis of the gland structure itself, and lastly in scirrhoid induration, the tissue of the duct becomes involved, the vital contractility is lost, dilatation ensues with stagnation of the secretion,[1] and the same effect is brought about in an entirely different manner. Instead of dilatations and cysts being the result of duct occlusion, by a stone already formed, they have here become the cause of its formation, and yet it is in some cases most difficult to say, from a study of pathological appearances, which is the cause, and which the effect.[2]

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[1] Rokitansky, A Manual of Pathological Anatomy, Syd. Soc., London, 1849: Abnormities of the different ducts and their contents, vol. ii. § 2, p. 180.......Portal, Cours d'anat. méd. Paris, 1803; Pancréas engorgé par des concrétions pieurreuses, t. v. p. 356.......Salmade, Recueil périodique de la Société de Médecine de Paris, 1797–98: L'observation à l'histoire des anévrismes, tome iii. p. 454.......Dufrèsne, Traité de l'affection calculeuse du foie et du pancreas, Paris, 1851: Article deuxième, p. 494....... Schupman, Hufeland's Journal, 1841, iii. Geschichte einer scirrhösen Hypertrophie der Leber und des Pankreas, etc., xcii. Bd., s. 41.......Wilson, loc. cit.......Curnow, Trans. Path. Soc., London, 1873, 23: Pancreas with numerous calculi in its ducts, vol. xxiv. p. 136.......Muhlbauer, Neue Notizen (von Froriep) Weimer, 1822–1847.......Devilliers, Revue médicale, Décembre, Paris, 1844, tome iii. p. 576.......Eller, Collection Académique, Paris, 1755. Recherches sur la formation des pierres ou concrétions, gravalleuses dans le corps humain, etc. Partie Etrangère, tome x. p. 85.......Sandifort, Observationes anatomico-pathologicæ, Lugd. Batav., 1777. Lib. iii. cap. iv. p. 73....... Virchow, loc. cit.

[2] Cawley, London Medical Journal, 1788: A Surgical Case of Diabetes, etc., vol. ix. part iv. p. 286; also Sammlung Auserlerner abhand., Leipsig, 1789, 13 Bd., s. 112....... De Graaf, Tractat anat. med. de succi pancreatici natura et usu., Ludg. Batav., 1671, cap. vii. p. 112.......Lieutaud, Hist. Anat. Med., Paris, 1767, liber i. § viii. obs. 1045, p. 244.......Janeway, New York Medical Record, 1872: A case of Calculi of the Pancreas, etc., vol. vii. p. 357.

[3] Rokitansky, ibid.

[4] Curnow, ibid.......Friedreich, op. cit. p. 615, and Calculi, p. 618.......Klebs, op. cit. s. 547.......Parsons, British Medical Journal, 1857, vol. i. p. 475.

[5] Recklinghausen, op. cit. b. Concretionen, Ektasien des Ductus Veröderung des Parenchyms des Pankreas.

[6] Delafield, op. cit. p. 202.......Friedreich, op. cit. p. 615.......Parsons, loc. cit.

[7] Klebs, op. cit. s. 544.......Delafield, op. cit. p. 203.

It is clear, then, that an obstruction to the free outflow of the pancreatic secretion will cause retention and accumulation of that fluid within the ducts of the gland, and thus favour the precipitation of its inorganic constituents. But it is possible that the secretion may become so altered in the nature or relation of its chemical components, as to bring about a deposition of its inorganic elements, without any preliminary obstruction or dilatation of the gland ducts. From a study of the reported cases of calculous disease of the pancreas, it will be seen that in many instances calculi have been found within the gland *where no obstruction of the ducts had existed*, such as described in the foregoing and succeeding pages.[3] It now, therefore, remains to be seen what are the changes in the pancreatic secretion which result in the formation of calculi, and how these changes are brought about. By a comparison of the chemical constitution of the normal pancreatic juice with that of pancreatic calculi, one will be struck by the following facts: In three analyses given of the former, lime, whether in combination with organic matter, or as free phosphate of lime, is present only in very small amount, or, as in a fourth analysis, is not mentioned at all; whereas, in the various chemical examinations made of the latter, lime, both as a phosphate and as a carbonate (an element not found at all in normal pancreatic juice), is present in very large amount, and forms either the entire calculus,[1] or else by far the greatest part of it.[2] In addition to the carbonate of lime already mentioned, three other inorganic substances have been found in pancreatic calculi, which, according to the analysis above referred to, have no place in the pancreatic juice; they are the chlorate of sodium,[3] carbonate of magnesium,[4] and oxalate of lime.[5]

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[1] Rokitansky, loc. cit.

[2] Gould, Catalogue of the Anatomical Museum of the Boston Society for Medical Improvement, Boston, 1847: Several pancreatic calculi, extensive disease of the pancreas, §§ 575-6, p. 173.......Dufrèsne, loc. cit.

[3] Matani, loc. cit.......Baillie, The Morbid Anatomy of the Human Body, London, 2d ed. 1812, p. 115, and, London, 1833, Calculi of the Pancreas, p. 222.......Elliotson, The Principles and Practice of Medicine, 2d ed., London, 1846: Diseases of the Pancreas, p. 1009; also, Medico-Chirurg. Trans., London, 1833, on The Discharge of Fatty Matters from the Alimentary Canal and Urinary Passages, vol. xviii. p. 67.......Bonetus, vide Bigsby, loc. cit.......Gould, op. cit. § 575.......Clayton, Medical Times, London, 1849: A case of Calculi of the Pancreas, etc., vol. xx. p. 37.......Henry, loc. cit....... McCready, New York Journal of Medicine, 1856: A case of Pancreatic Calculi, in Proceedings of Patholog. Soc. p. 78.......Recklinghausen, op. cit. 1, a. p. 360.......Cornil et Ranvier, loc. cit.......Cornil and Ranvier, trans. loc. cit...... Schmitt, loc. cit.

In conclusion, let us bear in mind the close analogy which exists between the pancreas and its kindred salivary glands, in both of which calculi are found, produced by the same general causes, and followed by the same effects.[6] How does the chemical composition of salivary calculi compare with that of the salivary fluid? It has been remarked, that the saliva from which salivary calculi are formed, must be in an unhealthy state, for, while these concretions consist chiefly of phosphate of lime, sometimes containing as much as ninety-four per cent. of that salt, little, if any, is to be found in the normal salivary secretion.[7] From this it is clear, that in true salivary calculi, as in those of pancreatic origin, the quantity of the salts of lime is much greater than in the fluids secreted by these glands. The means, by which the pancreatic secretion is so changed inside the body as to bring about a precipitation of its inorganic constituents, must still remain a matter of doubt. Various explanations have been advanced, but none are entirely satisfactory. It is possible that physiological variations are occasionally present in the composition of the pancreatic juice, as in the secretion of other salivary glands, in which occur temporary changes in quantity, in colour, in consistency, and, as indicated by the taste and alkaline or acid reaction, in chemical constitution.[1] Further, local changes in the tissue of the secreting portion of the pancreas,[2] or in that of its ducts, may produce such an influence upon the secretion as would lead to the crystallizing out of its less soluble salts. It has been said that the excess of phosphate of lime present in calculi is most probably caused by an irritation or inflammation of the mucous membrane lining the ducts,[3] and if this be true, there can be but little doubt that an excess of this phosphatic salt, which, as we have seen, is present in nearly all pancreatic calculi, can be formed in like manner in the ducts of the pancreas.

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[1] See in Table of Free Concretions. Baillie, Gould, and Collard: Henry, La France Méd. et Pharm., op. cit. p. 42.......Pemberton, on Various Diseases of the Abdominal Viscera, Richmond, Va., 1830: chap. iv. The Pancreas, p. 64.

[2] See in Table of Free Concretions: Clayton, Clark, McCready, Recklinghausen, Cornil and Ranvier, Janeway, Curnon; also, Wilson, loc. cit., Rokitansky, loc. cit.......Gross, Pathological Anatomy, Phila. 1845: Of the pancreas, chap. xxiv. p. 689.......Henry, La France Méd. et Pharm., loc. cit. p. 42.......Wollaston, see Pemberton and Henry, ibid.

[3] Henry, ibid. [4] Henry, Journal Méd. et Pharm., loc. cit.

[5] Golding Bird (See Wilks & Moxon, Lectures on Pathological Anatomy, Phila. 1875: Diseases of the Pancreas, Calculi, p. 470).

[6] De Graaf, loc. cit., Eller, loc. cit., Matani, loc. cit., Rokitansky, loc. cit., Henry, La France Méd. et. Pharm., loc. cit.; Friedreich, op. cit. p. 618; Wilks & Moxon, loc. cit., Portal, loc. cit.......Bécourt, Recherches sur le pancréas, Thèse, Strasbourg, 1830: Concrétions inorganiques, § 5, p. 69.......Mondière, Archives Générales de Médecine, ii. Série, Paris, 1836: Recherches pour servir à l'histoire pathologique du pancréas, tome xii. p. 147.......Bigsby, Edinburgh Medical Journal, 1835: Observations on Diseases of the Pancreas, vol. xliv. p. 97.......R. D., Dictionnaire de Médecine, Paris, 1841: Maladies du Pancréas, concrétions osseo-pieurreuses, tome xxiii. p. 65.

[7] Jones & Sieveking, A Manual of Pathological Anatomy, Phila. 1854: Abnormal conditions of the pancreas and other salivary glands, p. 532.

It seems proper, before leaving this part of our subject, to record in brief the chemical analyses made of two pancreatic calculi, which, for obvious reasons, are not to be found in the list of free concretions,[4] with the analyses of two salivary calculi, in order to illustrate the close analogy which is said to exist between pancreatic and the so-called salivary calculi.

1.[5] A pancreatic calculus, wt. 9.06 gm., contained—

Phosphate of lime	72.30
Carbonate of lime	18.90
Phosphate of soda	traces
Chlorate of sodium	"
Animal matter	8.80
In	100.00 parts.

2.[6] A pancreatic calculus found in canal of Wirsung of an ox contained—

Carbonate of lime	91.65
Carbonate of magnesium	4.15
Organic matter	3.
Water	1.20
In	100.00 parts.

3.[7] A salivary calculus found in Steno's duct of a woman contained—

Phosphate of lime	55
Carbonate of lime	15
Animal matter	25
Oxide of iron	2
Magnesium	traces
Waste	3
In	100 parts.

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[1] Rokitansky, loc. cit.......Friedreich, op. cit. p. 618.

[2] Recklinghausen, op. cit. i. b.

[3] Recklinghausen, ibid.......Jones & Sieveking, loc. cit.

[4] Henry, Journal de Chêmie Médicale, op. cit. p. 273.

[5] Henry, Journal Méd. et Pharm., loc. cit.

[6] Schulze, Journal für p. Chemie, xxxix. p. 29: Rap. an de Berzelius, 1848, p. 412: cf. Henry, ibid.

[7] Lassaiqué, Traité de Chémie, tome ii. p. 614: cf. Henry, ibid.

4.[1] Another salivary calculus contained—

Phosphate of lime	75.
Carbonate of lime	20.
Animal matter Waste	5.
In	100 parts.

The changes in the parts or tissues adjacent to the pancreas, which are of force in the production of pancreatic calculi, are as numerous and as varied as those within the gland itself. Yet we will find the same thing here as there, different causes with like effects; an obstruction to the free escape of the pancreatic secretion, a stagnation, an inspissation, and the formation of a calculus, with all the train of phenomena attending thereon. And we repeat here what has been said in a former section, not only that calculi, by occluding the pancreatic ducts, cause their dilatation and the formation of retention cysts; but that these cysts, whether so formed or not, favour in their turn the production of calculi,[2] and that, therefore, whatever causes are assigned for the development of retention cysts can also be considered as secondarily productive of calculi. From a careful study of the cases of pancreatic calculi which we have collected, we find no instances in which the production of a concretion has been caused by compression of the excretory duct through changes outside of the gland; nevertheless, since such compression is possible, and may have been overlooked, we cannot refrain from briefly referring to some of the changes whereby it may be brought about. Among these may be mentioned peripancreatic induration and adhesions, especially near the head of the gland;[3] gall-stones lodged in the common duct and pressing upon the duct of Wirsung;[4] a cancerous growth in the duodenum;[5] and, finally, changes, as the result of which the pancreas becomes displaced.[6]

4. *Mode of Escape of Pancreatic Calculi.*—Pancreatic calculi may escape into the duodenum, accompanied by a paroxysm of pain or colic, similar to that which attends the passage of biliary or of renal calculi.[7] This, instead of being only a probable method of escape,[8] is, we believe, not at all infrequent.[9] No case in which a direct observation has been made can, however, be brought forward in proof of this assertion. As will be

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[1] Lecannau, Journ. de Pharm. et de Chir., Décembre, 1827, p. 626.

[2] Virchow, Die Krankhaften Geschwülste, Berlin, 1863, i. Bd., s. 276.......Friedreich, op. cit. p. 615.

[3] Hoppe, Virchow's Archiv, 1857, xi. Bd. s. 96: cf. Friedreich, ibid.

[4] Engel, Oesterreich med. Jahrbücher, 1841: xxiii. u. xxiv. Bd.

[5] Friedreich, op. cit. pp. 601–15.

[6] Engel, ibid; Friedreich, ibid.

[7] Friedreich, op. cit. p. 618.

[8] Ancelet, Essai Analytique sur l'anatomie pathologique du Pancréas, Thèse, Paris, 1856, tome i. p. 27.

[9] Pemberton, loc. cit., Portal, loc. cit.: cf. Dufrêsne.......Schupman, loc. cit., Wilson, loc. cit., Clayton, loc. cit.

discussed in a later section, it is almost impossible to tell from symptoms whether a pancreatic or biliary calculus is being passed, nor is it easy to recognize pancreatic concretions in the stools. It is, nevertheless, possible that in many of the cases which we have collected where, during life, pain, more or less characteristic, was present, and after death pancreatic, but no biliary calculi, were found, nature had sought this mode of relief.[1]

It is known that calculi, acting as foreign bodies within the pancreas, sometimes give rise to inflammation and ulceration, with subsequent perforation of the gland substance. In one reported case perforation was found at the post-mortem examination, and, although several calculi had remained in the pancreas, one had escaped into the abdominal cavity. The patient's death was attended with symptoms denoting internal hemorrhage, and a large quantity of coagulated blood was found within the peritoneal sac.[2] In another case, the record of which is rather obscure, one concretion was found in the pancreas, and several others in an abscess, situated in the mesentery and opposite the point (the right lumbar region) where, during life, the patient suffered the greatest pain.[3] Although no lesion of the pancreas was noted, it is not unlikely that the calculi escaped in the same manner as is mentioned in the last case. A later writer describes this abscess as peri-pancreatic, but we do not think that the original account warrants this construction.[4]

5. *Results following the Impaction of Calculi in the Ducts of the Pancreas.*

a. Dilatation of ducts and formation of cysts.
Hemorrhagic cysts.
Hemorrhage into the pancreas.

Dilatation of the ducts of the pancreas, with or without the subsequent development of cysts, is one of the results which most often follow the formation of calculi within the gland.[5] Virchow, having in mind the close analogy (already pointed out) which exists between the pancreas and the salivary glands of the mouth, has given the name Ranula Pancreatica to all dilatations of the pancreatic ducts leading to the formation ot cysts.[1] This dilatation of the ducts of the pancreas is due to a distension of their walls by the mechanical pressure of a retained secretion. The degree of dilatation, other things being equal, is in direct proportion to the completeness of the obstruction to the outflow of the pancreatic juice. Although a calculous concretion is one of the usual and most effective causes of this obstruction, nevertheless many other changes, both within and without the gland, undoubtedly produce the same effect. These alterations have been already briefly referred to,[2] but many others exist which it is not our intention at present to describe.[3] It suffices to say, given an impediment, complete or partial, to the free escape of the pancreatic secretion, and the most natural result is a mechanical distension and dilatation of the ducts behind the obstructed point.

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[1] Wilson, loc. cit.......Mercklin, Ephem. nat. cur. decur., Frankfort und Leipsig, 1678: De ingen. calc. in mesen. et pan. repert, Ann. 8, obs. 50, p. 78.......Galiati, De morbus duobus observ. 1758, p. 26; or Comment. Bonon, tome iv. p. 34. (NOTE.—It has been impossible to gain access to these two works. The case is quoted by Sandifort, Mondière, R. D., Bècourt, and by others.) Elliotson, Med. Chirurg. Trans., loc. cit.......Schupman, loc. cit., Gould, loc. cit., Clayton, loc. cit., Dufrèsne, loc. cit. ......Clark, London Lancet, Aug. 16, 1851: A case of disease of the pancreas, etc....... Fournier, Anc. Journal, tome xlv. p. 149; or, more correctly, the journal being better known under its more recent name, Journal de médecine, chirurgie, pharmacie, Paris, 1776, tome xlv. p. 149.

[2] Clayton, loc. cit.

[3] Mercklin, ibid.

[4] Klebs, op. cit. s. 544.

[5] Matani, Baillie, Schupman, Gould, Rokitansky, Dufrèsne, Clark, Virchow (Verhand. der Med. physik. iii.), Ancelet, Recklinghausen, Cornil & Ranvier, Janeway, Curnow, Fournier, Meckel and Delafield.

Among the cases of pancreatic calculi collected, dilatatión of the main duct is the result of obstruction the most frequently observed. Generally, this dilatation represents only an exaggeration of the normal calibre of the duct, which throughout the greater part of its length is more or less uniformly dilated.[4] If, however, the obstruction is near the point of exit into the duodenum, and is complete and continued, the dilatation does not cease here, but the accessory ducts in their turn become enlarged,[5] presenting the appearance of diverticula, opening into the duct of Wirsung, and separated from one another by valvular folds or transverse ridges, formed by the coats of the ducts, which may be either thickened or attenuated.[6] Again, instead of these close-set expansions, there may exist at intervals single fusiform dilatations.[7] It is possible for the accessory ducts to undergo dilatation without the involvement of the main duct. Thus they may become closed at their exit by the presence of a calculus, by local interstitial inflammatory thickening, or by catarrhal swelling of their lining membrane, with or without the collection of a stringy catarrhal secretion, the main duct remaining patent throughout. The degree of dilatation, depending as it does upon the conditions already pointed out, varies considerably in different cases. We have the size of the dilated main duct compared to that of a quill,[8] of the thumb,[9] it is said to be dilated,[1] much dilated,[2] or prodigiously dilated.[3] In one case only was an accurate measurement made, when the diameter of the duct was found to be one inch.[4]

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[1] Virchow, Die Krankhaften Geschwülste, loc. cit.......Parsons, British Medical Journal, June 15, 1857, vol. i. p. 475.

[2] Reference may here be made to the section on the Mode of Origin of Pancreatic Concretions.

[3] Klebs, loc. cit.: Cysts, p. 547.......Friedreich, loc. cit.: Cysts, pp. 600–15.......Parsons, loc. cit..... Cornil & Ranvier, loc. cit.: Kystes, p. 974 : Trans. Cyst., p. 581....... Virchow, loc. cit.......Ancelet, loc. cit.: Kystes, p. 26.......Rokitansky, loc. cit.......Cruveilhier, Traité d'anatomie pathologique générale, Paris, 1856 : Kystes Pancréatiques, tome iii. p. 365.

[4] Klebs, op. cit. s. 547.......Meckel, Baillie, Gould (op. cit., § 575), Rokitansky, Virchow, Ancelet, Recklinghausen, and Cornil et Ranvier.

[5] Matani, Janeway, and Curnow.

[6] Klebs, Rokitansky, Friedreich, and Recklinghausen.

[7] Rokitansky. [8] Rokitansky. [9] Matani.

The study of the cysts, or, as the Germans are pleased to call them, *Ektasien*, resulting from an extensive and continuous impediment to the outflow of the pancreatic juice, is an extremely interesting one, and will be briefly alluded to as one of the many results following the impaction of concrements. All such cysts found in the pancreas belong purely to the class of retention cysts, and although in some cases extensive secondary changes seem to render their origin doubtful, yet it is comparatively easy to exclude pouches beginning outside of the pancreas, even though pushing aside its glandular tissue, and entering into communication with its duct, aneurismal dilatations of arteries in the gland, and, finally, cysts originating from entozoic formation.[5]

Cysts of the pancreas present many varieties in form and size, have been studied with some care by pathologists, and are of the utmost symptomatological importance. Two distinct classes are met with: those which emanate in an extreme degree of dilatation of the main duct of the pancreas, and the small multiple cysts originating from a similar dilatation of the small ducts within the acini, or of the terminal vesicles of the gland. The former sometimes attain an immense size, in one case measuring three by four inches;[6] in others compared to the size of a child's head.[7] Their form is generally spherical or oval,[8] but varies extensively in different cases.[9]

Whereas, in ordinary dilatation of the main or of the accessory ducts of the pancreas, and also in the first period of the history or development of a cyst, there is a simple retention of the secretion without any notable alteration in the walls of the duct or cyst, or in the secreting structure of the gland itself, yet, in the second period, in the *kyste confirmé*, changes occur which cannot be passed over unnoticed. Here the cyst walls become thick, fibrous, tough, cartilaginous, or even ossified. Their internal surface may be smooth, or may present changes similar to those which occur on the internal surface of arteries, namely, fatty or chalky precipitations or aggregations, such as are seen in the later stages of endo-arteritis. In other instances the walls of very large or old cysts become the seat of albuminoid or of purulent infiltration. The mischief does not however stop here; the constantly enlarging cyst, with its thickened and hardened walls, encroaches upon the substance of the gland itself, and the result of this pressure is seen in morbid alterations of the gland parenchyma. Through chronic interstitial growth and induration the entire gland structure, even to its last acinus, atrophies, loses its function, and is completely destroyed, or again is entirely replaced by adipose tissue.

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[1] Ancelet, Janeway. [2] Curnow. [3] Baillie.

[4] Gould, op. cit. § 575.

[5] Recklinghausen, op. cit. i. a. [6] Gould.

[7] Recklinghausen, Ancelet, Bécourt......Duponchel, Soc. Méd. d'Émulation, Paris, 1824, tome ix. p. 76.

[8] Recklinghausen, Gould. [9] Krebs, Friedreich, etc.

The nature of the contents of pancreatic cysts varies so constantly that it would be impossible to embody in any one description all of the peculiarities met with. In some cases cysts were found filled with the altered secretion of the gland; in others, with a pure mucus; again, with a serous fluid; but, in a large proportion, products of retrograde tissue metamorphosis, broken-down cells, cholesterin crystal, purulent matter, or blood extravasations, with crystals of hæmatoidin, were present.[1]

Lastly, as has been already mentioned, calculous concretions are sometimes formed within old pancreatic cysts. Under the head of Acne Pancreatica, the contents of small cysts have been described at sufficient length in a former section.

The quantity of fluid present in a cyst will, of course, vary with its size; the largest amount of which we have record was between ten and fourteen ounces.

Many and varied are the changes induced in the surrounding tissues and organs by the presence of pancreatic cysts; in fact, the contents of both the epigastric and hypochondriac areas may become more or less involved by the proximity of the diseased structures, and present the most remarkable variety of pathological processes. Those which most properly concern us are such as will, if accurately understood, be of use in enabling us to recognize during life the seat and nature of the disease. These lesions are, as a rule, simply the result of pressure, and will necessarily vary with the size and situation of the tumour. And, again, as an aneurism of the thoracic aorta, by pressing upon the trachea, without necessarily causing any alteration in its structure, will produce marked if not fatal symptoms; so a cyst of the pancreas, by mechanical pressure alone, will endanger if not end life. The changes which are ordinarily found consist, in the majority of cases, in the formation of morbid adhesions, from which the most singular distortions result. None of the contents of these regions escape implication. The portal vein,[2] inferior cava,[3] and splenic vein;[4] the pylorus,[5] duodenum,[6] colon,[7] gall-bladder, and biliary passages;[8] the stomach,[9] liver,[10] spleen,[11] and right kidney;[12] and, lastly, the solar plexus of nerves;[13] may all be structurally involved or locally displaced, rendering diagnosis most difficult, and post-mortem examination most unsatisfactory.

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[1] Pepper, Centralblatt für die Med. Wissench. 1871, p. 156.

[2] Dufrèsne, Recklinghausen.

[3] Dufrèsne, Klebs.

[4] Recklinghausen.

[5] Fournier.

[6] Gould, Dufrèsne, Fournier.

[7] Fournier, Parsons.

[8] Dufrèsne, Fournier.

[9] Gould, Recklinghausen.

[10] Gould.

[11] Recklinghausen.

[12] Fournier.

[13] Recklinghausen.

In the positions occupied by cystic enlargements of the pancreas, there is not enough similarity to enable us to deduce any clinical laws. Thus the tumour may be situated below the right lobe of the liver, and between the intestines and the posterior wall of the abdomen,[1] at the entrance of the common duct into the duodenum,[2] between the left end of the stomach and the upper end of the spleen,[3] midway in the pancreas,[4] or in the left hypochondrium.[5] Finally, with regard to their termination, large pancreatic cysts may rupture, their contents being discharged into the duodenum, stomach, or peritoneal cavity, and so cause death.[6]

Before closing our remarks upon cysts of the pancreas, the subject of hemorrhagic cysts is deserving of notice, and with it will be incorporated a short account of hemorrhages into the pancreas, for, owing to the close analogy which exists between these two conditions, it is thought desirable to discuss them together.

Old pancreatic cysts frequently contain more or less blood (generally disorganized, and of a chocolate-brown colour), which, if coagulated, constitutes the so-called hæmatoma.[7] On the other hand, fresh, red extravasations are occasionally met with. It is necessary, in speaking of hemorrhage proper, to distinguish between that form which results from a passive congestion of the gland, dependent upon some cen ral obstructive disease, along with chronic inflammatory changes in the vascular system,[8] and hemorrhages which take place into the gland, the result of the rupture of vessels which have undergone a primary fatty degeneration.[9] These constitute the so-called apoplectiform hemorrhages into the pancreas. The second of these conditions is admirably illustrated by three cases, the report of which was published in a foreign journal a few years since.[10] In the first the individual was apparently in perfect health; in the second, an epileptic, otherwise healthy; in the third, a drunkard. They were all quite corpulent. In each case death occurred suddenly, and without apparent cause, while after death extensive fatty degeneration of the pancreas was observed. The hemorrhage in one case was more of the nature of a bloody infiltration of the gland substance. In another the infiltration was also observed around the gland; while in the third there was a somewhat extensive hemorrhage into the pancreas. In two of these cases there was also an effusion of blood into the duodenum. Since the quantity of extravasated blood was in no case sufficiently large to bring about speedy dissolution from hemorrhage, *i. e.*, direct loss of blood; and since in each case great hyperæmia of the semilunar ganglion was noticed, without structural alteration in nerve cells or fibrillæ, the cause of instantaneous death is explained on the theory, that the suddenly enlarged pancreas, by pressure on the semilunar ganglion, and solar plexus of nerves, produced an immediate arrest of the heart, by a reflex inhibition exerted through the vagus nerve, as in Goltz's familiar experiment with the frog.

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[1] Gould.
[2] Dufrèsne.
[3] Clark.
[4] Recklinghausen.
[5] Parsons.
[6] Friedreich, Pepper, Klebs.
[7] Klebs, Ancelet, ann. med. Störk, Vin. Wien, 1757, Gould, Duponchel, Clark, Pepper; Gros, Archives Gén. de Méd., Paris, 1849, IVe série, vol. xix. p. 215.
[8] Friedreich, Störk; Rugg, London Lancet, May 18, 1850: Fatal Hemorrhage from Pancreas.
[9] Friedreich.
[10] Zenker, Tagblatt der 47 Versammlung deutsches Naturforscher und Aerzte in Breslau, 1874, s. 211.

6. *Inflammatory Changes.*—Of those secondary conditions which are the result of the presence of calculi within the pancreas, duct dilatation and cyst formation are certainly the most interesting when viewed from a pathological standpoint; and, moreover, when excessive, by revealing to the diagnostician the presence of a local swelling, serve to explain many obscure and intractable symptoms. But, while acknowledging the greater interest of that part of our subject, it is necessary to enumerate at least the alterations of structure which result when the pancreas becomes the seat of stony concretions. These are in the main similar, when considered as mere pathological processes, to changes occurring in any other gland, and depend primarily upon inflammation. The pancreas may thus become the subject of acute, subacute, or chronic inflammatory processes; those which to us are of the greatest interest being the acute, suppurative, and the chronic interstitial inflammations. There is no doubt that the duct-wall first becomes the seat of change, and a condition is described in which it was found thickened, through an increase in the surrounding connective-tissue substance, its lining cells having undergone marked alterations.[1] No accurate description of duct inflammation resulting from the irritation produced by a calculus is, however, given by systematic writers. Dr. Roland G. Curtin, of Philadelphia, has reported a case of what he believes to have been a primary catarrhal inflammation of the ducts of the pancreas.[2] The microscopic examination of the specimen showed a condition of things not very dissimilar to the description just given. Dr. Curtin suggests, however, that the inflammatory process may have begun in the duodenum, and from thence involved the pancreatic duct secondarily, this theory being borne out by the fact that during life symptoms of intestinal derangement preceded any evidences of pancreatic obstruction. Whether a primary catarrhal inflammation of the pancreatic ducts is possible, must of course remain an open question; but that such an inflammation, whether primary or secondary, will give rise to subsequent changes in the pancreas, should it affect the duodenal end of the duct of Wirsung

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[1] Cornil and Ranvier, loc. cit.

[2] A Contribution to the Pathology and Therapeutics of the Pancreas. A paper read before the Pennsylvania State Medical Society, 1881, by Dr. Roland G. Curtin, of Philadelphia.

alone, or be confined to the smaller branch ducts, is a fact proven by the case of Dr. Curtin, and by the results recorded by other investigators.

It is possible, though of rare occurrence, to have an inflammation accompanied by deposits of purulent matter in the gland structure. We have been able to collect but three cases in which this condition was the result of the presence of calculi. In one, the head of the pancreas was merely touched with points of ulceration;[1] in a second, an abscess of the size of a nut, containing much purulent matter and many calculi, was found in the centre of the gland;[2] while in a third, a very large abscess, containing calculi, and having for its walls the dilated head of the gland, was discovered.[3] Such abscesses may open in any direction; the second referred to discharged a part of its contents into the stomach through a hole the size of a thumb.

A chronic interstitial inflammation of the pancreas is described, which by hyperplasia of connective tissue may lead to hypertrophy, atrophy, induration, and fatty degeneration. The condition of hypertrophic cirrhosis is rare. Atrophic cirrhosis is common, and the induration accompanying it is marked. This induration, which may give to the gland a feeling of cartilaginous hardness, has very often been confounded with scirrhus, so that in many cases of supposed cancerous disease, there is no doubt that the real lesion was one of cartilaginous induration.[4]

a. Fatty Changes.—In speaking of fatty disease of the pancreas, a distinction must be drawn between the condition known as lipomatosis and the true fatty degeneration, or *transformation graisseuse.* In the first of these, fat develops in the interacinous connective tissue, or else on the periphery of the gland, pushes its way in, and produces atrophy and even complete destruction of its proper secreting structure, the whole gland being converted into a mass of fat.[5] The second is a true fatty metamorphosis, beginning in the gland cells, and leading to complete destruction of the acini and subsequent atrophy of the whole viscus.[6] These two conditions are to a certain extent inseparable, and since no microscopic examination was made, it is impossible to say whether, in the two reported cases of pancreatic calculi in which fatty changes in the gland were the result, fatty infiltration or fatty degeneration existed alone or together.[7]

[1] Dufrèsne. [2] Muhlbauer. [3] Fournier.

[4] Cawley, Salmade, Schupman, Friedreich, and Klebs.

[5] Maier, Archiv der Heilkunde, Leipsig, 1865; Fall von ausgezeichnete (r) Verfetlung der Pankreas, s. 168.......Klebs, op. cit. s. 562.......Friedreich, op. cit. p. 624. ......Cornil et Ranvier, op. cit. tome ii. p. 970: Dégénérescence graisseuse.......Bécourt, op. cit. p. 50.

[6] Jones, Med.-Chirurg. Trans., London, 1855: Observations respecting Degeneration of the Pancreas, p. 195.......Salter, Encyclopædia of Anatomy and Physiology, Part XLIV.......Rokitansky, Lehrbuch der Patholog. Anat., 1861, iii. s. 313, 369.......Bécourt, op. cit. p. 50.......Ancelet, op. cit. p. 29.......Bock, Lehrbuch der Pathol. Anat., Leipsig, 1847: Pankreas, § 7, s. 669.......Cruveilhier, loc. cit.

[7] Clark, loc. cit., Recklinghausen, op. cit. 16.

II. SYMPTOMATOLOGY.

In endeavouring to inform ourselves with regard to the symptomatology of calculous disease of the pancreas, the same difficulties are encountered as beset our studies of its pathological anatomy. The older writers are as obscure in their description, and brief in their enumeration of symptoms, as they are obscure and brief when pathological conditions were to be described or enumerated. And more, in modern times, instances of calculous disease of this gland are rather regarded as curiosities to be alluded to by a pathological anatomist, than as subjects worthy the study of a clinician. And considering, as we must, the great number and variety of those diseases with which the active practitioner daily comes in contact, and which still remain so intractable to all therapeutic resource, we can well understand how a condition, relatively so rare as calculous disease of the pancreas, a condition about which we can learn so little from literature, either past or contemporary, whose history and causation are so obscure, whose diagnosis is so difficult, and whose treatment is so ineffectual, should fail to receive more than superficial study, or passing mention. Therefore, when, as here, we leave aside the study of the lesion, its causation, and its treatment, and confine ourselves solely to those signs and symptoms which would lead us to infer its presence, we cannot hope to find anything so characteristic as is revealed by the physical exploration of the chest, or the temperature curve of a fever.

The pancreas, it is true, plays a most important part in the great act of digestion, but when, by disease, its functions are perverted or destroyed, so intimate are both its structure and functional connection with the other members of the same great system, and so great may be its pathological alterations, without there being any tangible evidence of the same, that from the start our diagnosis becomes most difficult. But even should disease be located in the pancreas, it would be almost, if not completely impossible, to differentiate, for example, between cancerous and calculous disease of that organ. It will be our endeavour, in the following pages, to make, from a study of the cases at our command, a complete, but we fear an unsatisfactory recapitulation of the various views advanced from time to time upon the symptomatology of this disease, and to give due weight to such as seem to us of the greatest clinical importance, both in pure, uncomplicated cases of pancreatic calculi, and in those accompanied by disease of other organs and tissues.

It is most important, while endeavouring to explain the symptoms of calculous disease of the pancreatic ducts from a pathological standpoint, to remember two things, already briefly referred to, namely, the anatomical position and local relations of the pancreas, and its physiological function as a most important digestive organ. Deep-seated and surrounded as it is by structures whose organic and functional integrity is of such importance to the well-being of the economy, it is easy to see how, for example, from mere enlargement, by mechanical pressure, it could cause changes and disturbances the most grave. In one case, a pancreas, three times its normal size, and filled with concretions, so pressed upon the aorta as to produce an enormous aneurismal dilatation of that vessel above the point of obstruction.[1] In another, the return flow of blood through the inferior cava was interfered with to such an extent as to cause ascites with œdema of both legs.[2]

In a third, where the spleen was found much softened and disintegrated, in default of a more suitable explanation, the lesion is attributed to compression of the splenic artery and veins by the enlarged, earthy, and indurated pancreas;[3] while, from pressure upon the excretory ducts of the liver, we find jaundice from retention, a condition by no means uncommon.[4] When we consider, finally, the local relation between the pancreas and the cœliac plexus of nerves, we find ourselves entering upon a branch of our subject the most interesting.

In a large number of the reported cases of pancreatic calculi, *pain* is mentioned as one of the most constant, and, at the same time, one of thè most annoying of symptoms. But it is not one kind of pain only that we find described, but rather two, differing essentially in character one from the other, and having, in most cases, two entirely distinct modes of causation. The one dull, heavy, a sense of weight, a sense of uneasiness, located in the epigastrium, lasting, in some cases, throughout the entire attack,[5] while, in others, it preceded the actual illness of the patient by many years.[6] The other, sharp, severe, sudden in its onset, irregular in its accession, spontaneously relieved, yet reoccurring when least expected.[7] Separately or together these two kinds of pain, in different degrees of severity, occur very often in cases of calculous disease, and, in seeking for their mode of causation, two conditions are, we think, necessary to afford a satisfactory explanation. The various sensations at first referred to, varying in degree from a feeling of uneasiness or of weight, to pain, generally constant, and, in some instances, quite severe, are due, it would appear, to the pressure of a hard and enlarged pancreas upon the cœliac plexus of nerves. Although it would seem, at first sight, as if the mere presence of calculi within the pancreas would be quite sufficient to account for these subjective symptoms, yet, as it has been remarked, the relative insensibility of the excretory ducts of this gland, as compared with those of the liver or kidney, renders it improbable that much inconvenience or pain would be caused, even though they were distended by calculous concretions.[1] Whereas, if the cœliac plexus could be so compressed (under circumstances such as we have above described) as to suffer atrophy, and even complete destruction of its ganglionic cells,[2] it seems very probable that this, or even a far less degree of pressure, would produce pain. Our explanation is further borne out by the fact, that, when the recumbent posture is assumed, the pain grows worse,[3] while occasionally it radiates to the back, chest, and even right shoulder.[4] We are, therefore, led to conclude that this symptom, pain, is the result of pressure, and may, as has been suggested, be called a cœliac neuralgia.[5]

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[1] Salmade, loc. cit.......Portal, loc. cit.......Dufrèsne, loc. cit.

[2] Dufrèsne, quoting Portal's second case, see Maladie du foie, obs. f, p. 300...... Clark, loc. cit.......Muhlbauer, loc. cit., noticed œdema of extremities of left side only.

[3] Wilson, loc. cit.

[4] Meckel, loc. cit.......Fournier, loc. cit.......Dufrèsne, ibid.......Gould, op. cit. § 576. ......Henry, loc. cit.......Friedreich, op. cit. p. 618.......Galiati, loc. cit.

[5] Mercklin, loc. cit.......Wilson, loc. cit.......Clayton, loc. cit.......Muhlbauer, loc. cit.......Fournier, loc. cit.......Henry, loc. cit.......Gould, op. cit. § 576.......Elliotson, Med.-Chirurg. Trans., loc. cit.

[6] Mercklin, ibid.......Gould, ibid.......Henry, ibid.......Galiati, ibid.......Pepper, Medical Ward Notes, Hospital of the Univ. of Pa., 1880.

[7] Schupmann, loc. cit.......Wilson, loc. cit.......Gould, loc. cit.......Clayton, loc. cit. ......Dufrèsne, loc. cit.......Clark, loc. cit.......Fournier, loc. cit.......Pepper, loc. cit.

But when we come to consider the other kind of pain described, another and more suitable explanation offers itself. This, the true pancreatic colic, the result of the rapid distension of the duct of the gland by a stone forcing its way into the duodenum, the walls of the duct spasmodically contracting upon it, has been referred to in a former section. From a diagnostic point of view this symptom has but little weight, for it is difficult to tell, from the character or location of the pain, whether a biliary or a pancreatic calculus is being passed; as a pancreatic calculus, in its passage through the last part of the duct of Wirsung, by pressure on the common duct, would produce jaundice as effectually as would one of biliary origin.[6]

Before leaving this part of our subject, it is well to refer briefly to the existence of *mellituria*, as a symptom of calculous disease of the pancreas, since it occurs with sufficient frequency to demand attention.[7] In so much obscurity is the morbid anatomy of diabetes mellitus involved, that it would be alike useless and impossible, within the limits of this paper, to go at length into the theories that have been advanced as to its causation. Suffice it to say that, whereas, in each and every organ of the body, the morbid change, of which the condition known as mellituria is the result, has been from time to time located, it seems highly improper that the pancreas, as one of the organs of importance, should not bear its part of the burden. In one of two[8] cases of calculous disease (where, during life, mellituria was present), in which a careful examination of the cœliac plexus of nerves was made after death, changes consisting in the destruction of a certain number of its ganglionic cells could be observed. Now, since it is affirmed that extirpation or atrophy of the cœliac plexus will give rise to the presence of sugar in the urine,[1] it is plain how secondary changes in this plexus of nerves of sufficient gravity to bring about this condition, could be produced by the pressure of a pancreas, made large and hard by the presence of calculi. And, to go further, atrophy of the pancreas will follow these changes in the cœliac plexus as atrophy of the submaxillary gland will follow section of the vaso-motor nerves supplying it. The appearance of fatty stools after mellituria would seem to prove this.[2] There is no doubt at least that atrophy of the cœliac plexus will produce a vaso-motor paralysis throughout the whole area occupied by the pancreas, for all the vessels have been seen enormously dilated, and the spleen engorged and swollen.[3]

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[1] Gross, loc. cit.......Bigsby, loc. cit.

[2] Klebs, op. cit. s. 544.

[3] Wilson, loc. cit.......Gould, op. cit. § 576.

[4] Elliotson, Med. Ch. Trans., loc. cit.......Fournier, loc. cit.......Pepper, Med. Ward Notes, Univ. Hosp., loc. cit.

[5] Klebs, op. cit. s. 544.

[6] Friedreich, op. cit. p. 618.

[7] Recklinghausen, op. cit. I. a. and b.......Elliotson, Med. Chirurg. Trans., loc. cit....... Cawley, loc. cit.......Klebs, op. cit. s. 544.......Chopart, Maladies des voies urinaires. ......Cf. Klebs, loc. cit.......Pepper, Med. Ward Notes, Univ. Hosp., loc. cit.

[8] Klebs, op. cit. s. 544.......Recklinghausen, loc. cit.

In regard to those symptoms which are the result of an improper performance of the physiological functions of the gland, due to the presence of calculi within it, we can only enumerate, without endeavouring to explain, the phenomena mentioned in the reported cases. When the main excretory duct of the pancreas was entirely blocked up by concretions, the symptoms of digestive derangement were of course more marked than when the outflow of the pancreatic juice was in whole, or in part, permitted. In thirteen cases, we find the actual illness of the patient preceded for a variable length of time, and ushered in by more or less marked symptoms of gastro-intestinal derangement, accompanied in some cases by abdominal pain. During the progress of the disease, these symptoms become more persistent, and increased in severity, so that, in many cases, the termination of the organic disease in complete duct obstruction placed a limit to the patient's life, while, as has been remarked, we find changes in other parts of the body capable of spoiling life, but not of causing death.

In four cases, where, upon post-mortem examination, calculi were found in the pancreas, *vomiting* was one of the most distressing symptoms present during life.[4] In three others, bloody vomiting was noticed.[5] In six cases, *diarrhœa* was present;[6] in four, melæna;[7] while *constipation* was observed in six,[8] and fatty stools in three.[9]

The presence of fat in the stools is a symptom of great importance in the recognition of pancreatic disease,[10] but that it is not of absolute diagnostic value is proven by the well-known fact, that the same condition will follow upon the obstruction of the biliary passages. For, while the main action of the pancreatic juice upon fats is to cause their emulsification, this power is possessed, though to a less extent, by the bile, which, like the succus entericus, emulsifies fat, but does so to a degree entirely insufficient to meet the needs of the economy. Therefore, it must be remembered that not the bile alone, nor the pancreatic juice alone, can properly digest fats, but rather that they must act together, mutually aiding one another in the performance of this joint function. But since, as an emulsifying agent, the pancreatic juice is far more active than the bile, it might be argued that fatty stools would be more symptomatic of pancreatic than of hepatic disease. But, even should jaundice and fatty stools *both* be present in the same case, it by no means follows that they together point to biliary obstruction, for, as has been shown, a pancreatic calculus, lodged in the duct of Wirsung just as it enters the duodenum, may, by pressure on the common duct, prevent the escape of bile, and give rise to jaundice, while, at the same time, by this obstruction, but more by preventing the outflow of the pancreatic juice, cause the appearance of fat in the excreta. In proof of the first of these assertions, it may be said that in two instances of calculous disease in which the excreta presented a pale or clay-coloured appearance, upon post-mortem examination, no morbid alteration of sufficient gravity to cause this condition was found in the liver, and the dilated ducts contained no obstruction, the common duct opening freely into the duodenum, while the pancreatic duct was impervious, being blocked up with calculi.[1]

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[1] Klebs, op. cit. s. 544.

[2] Fles, cf. Klebs, op. cit. s. 544.

[3] Klebs, loc. cit.

[4] Cases reported by De Graaf, Mercklin, Galiati, and Schupmann.

[5] Wilson, Gould, Clayton.

[6] DeGraaf, Gould, Dufrèsne, Henry, McCready, Janeway.

[7] DeGraaf, Elliotson, Gould (two cases).

[8] Galiati, Cawley, Schupmann, Wilson, Clark, Wm. Pepper.

[9] Elliotson, Gould, Clark.

[10] This subject will be found discussed at length in the Medico-Chirurgical Trans., London, 1832, vol. xviii. p. 76, and elsewhere in the same volume by Lloyd & Bright.

It is strange that the presence of fat is noted in so few cases of this pancreatic disease, and, since the condition is one of some rarity, we will append an abstract of the three cases in which it was observed.

CASE I.[2] H. M., æt. 57; derangement of digestion for some years; fat noticed in urine, one month later also in excreta. In a short time fat oozed away from lower bowel without intermission or volition. Pain, progressive emaciation, and death from exhaustion. *Autopsy:* Liver congested, ducts dilated but healthy. Pancreatic duct at duodenal end completely blocked up by a mass of calculous matter.

CASE II.[3] W. P., æt. 45, had pain, diarrhœa, melæna; blood disappeared from, and fat appeared in stools and urine; amount of fat and severity of pain diminished as death approached; sugar appeared in urine; exhaustion, death. *Autopsy:* Liver, gall-bladder, and bile-ducts sound. Pancreatic duct and its larger branches crammed with calculi.

CASE III.[4] Man, æt. 40, had several attacks of hemorrhage from bowels; diarrhœa and constipation alternated; tenderness in epigastrium, pain; fat in stools (apparently only after articles containing fat had been eaten); no fat in urine; jaundice; death in coma. *Autopsy:* Liver rather small and dark-coloured; ducts dilated, contained no obstruction, free opening into common duct. Pancreatic duct obliterated at duodenal end.

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[1] Gould, op. cit. § 576...... Clark, loc. cit.

[2] Clark.

[3] Elliotson, Med.-Chir. Trans., loc. cit.

[4] Gould, § 576.

There have been discussed up to this point the symptoms which may be considered most characteristic of calculous disease of the pancreas, but there are certain others mentioned which it may be well to refer to. The appetite, "inordinate,"[1] "craving,"[2] "ravenous,"[3] especially in those cases where large quantities of fat passed away undigested, has been said to be impaired, tending in some cases toward complete anorexia.[4] As a natural result of the crippled condition of the nutritive processes, progressive emaciation,[5] accompanied by debility,[6] and followed by extreme prostration and exhaustion, would be expected.[7] Nothing worthy of comment is to be noticed with regard to the circulatory, respiratory, or nervous systems; whatever derangement we find mentioned can all be explained by the presence of other lesions, complicating the pancreatic disease. A most interesting condition is met with when we come to consider changes present in the urine. It will be seen, in the three cases already recorded, in which fat was found in the stools, that in two, fat passed away with the urinary secretion, and indeed, in one, made its appearance one month before it was detected in the excreta, and in such quantity as to float, when cool, in greasy cakes upon the surface.[8] Lastly, a peculiar colour of the skin, which is believed by some to be pathognomonic of pancreatic disease, must be mentioned, in connection with three cases of calculous affection in which it was observed. In them the appearance presented is variously described as being unhealthy, pale-yellow, dirty or earthy, and seems, except in one case, in which it was confined to the face, to have been general.

The ordinary methods of physical examination offer some partial assistance in the recognition of calculous disease of the pancreas. By palpation it is most difficult to recognize the position of this organ in health. "By deeply depressing the abdominal walls about a hand's-breadth below the umbilicus, by then rolling the subjacent parts under the hand (the stomach and colon must be empty), it might be possible to detect it in an individual who is thin and whose tissues are lax."[9] In a case where the size of the pancreas is only slightly increased by the presence of calculi, on careful palpation, it might be possible to detect a feeling of resistance, although when the gland is markedly enlarged, as in one of the reported cases of this affection (by an enormous stone formation situated in its tail, and the presence of a scirrhus at its head), a tumour of cartilaginous hardness might be perceived after careful examination of the abdomen. In the case referred to, the tumour was flat, slightly movable, and sensitive to the touch.[10] This is the only instance of this affection in which a local examination was made, except in three instances where a large cyst was the result of duct obstruction,[1] and in a fourth, where an enormous abscess, situated in the head of the gland and filled with calculi, caused the appearance of a localized epigastric swelling.[2] The diagnosis of abdominal tumours, as is well known, is extremely difficult, and is not a subject which it is our intention to discuss. To distinguish such a condition as we first described from a scirrhus involving the pyloric end of the stomach, or an induration of the left lobe of the liver, would, by physical exploration alone, be most difficult; while the characteristic thrill and bruit of an aneurism might be simulated by the rush of blood through an abdominal aorta compressed by an enlarged pancreas.[3] And so it is with cysts of this viscus, which can only be recognized by their situation, deep in the region of the pancreas, their round or oval shape, smooth surface, and the sense of fluctuation imparted to the touch,[4] while in both these conditions the general symptoms, which have been mentioned as more or less characteristic of calculous disease, would be of aid in determining upon a diagnosis.

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[1] Clark. [2] Gould. [3] Wm. Pepper.

[4] Schupmann.......Fournier. [5] Clayton, Clark, Wm. Pepper.

[6] De Graaf, Dufiésne, Clark. [7] Fournier.

[8] Clark, Wilson, Henry, Janeway.

[9] Sir Wm. Jenner, British Med. Journal, Jan. 16, 1869, p. 42. [10] Schupmann.

III. DURATION AND TERMINATION.

No decided laws can be laid down with regard to the duration and termination of this affection. So rare is it to find a pure uncomplicated case of calculous disease, that any generalization would be impossible; nevertheless, taking all those cases in which both clinical history and post-mortem examination are recorded, we find death from exhaustion or asthenia one of the most frequent of all terminations. In four cases, diabetes was the cause of death;[5] in one, rupture of an aneurism produced by the disease itself;[6] in another, internal hemorrhage, consequent upon a laceration of the pancreas, the calculus escaping into the peritoneal cavity.[7] Pneumonia is referred to in one case as hastening the fatal issue,[8] while aortic disease,[9] an accident of pregnancy,[10] and albuminuria and phlebitis[11] are each mentioned as having placed a limit to life.

BIBLIOGRAPHY.—*De Graaf*, Tractat. anat. med. de succi pancreat., Lugd. Batav., 1671, cap. vii. p. 112. *Mercklin*, Ephem. nat. cur. decur., Frankfort and Leipsig, 1678, Ann. 8, obs. 50, p. 78. *Eller*, Collection Académique, Paris, 1755, tome x. p. 85. *Meckel*, Koreff, Dissert. inaug. med. sistens theoret. consid. Icteri, Halæ magd., 1759, § 12, p. 16. *Störck*, Ann. med. Vindobon, 1760, t. ii. p. 245. *Matani*, Naturen geneeskundige Bibliotheek, gravenhage, 1765: and Gottingische anzeigen von Gelehrten sachen, 1765, No. 10. *Lieutaud*, Hist. anat. méd., Paris, 1767, liber 1, sect. viii. obs. 1045, p. 244. *Fournier*, Journ. de méd. chirurg. pharm., Paris, 1776, tome xlv. p. 149.

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[1] Gould, § 576.......Dufrèsne, quoting Portal's 2d case.

[2] Fournier.

[3] The diagnosis in such a case might be assisted, as suggested by Prof. Wm. Pepper, by placing the individual in the genu-pectoral position, and then using the method of palpation.

[4] Friedreich, Cysts, op. cit. p. 615.

[5] Cawley, Elliotson, Recklinghausen.

[6] Salmade.

[7] Clayton.

[8] Janeway.

[9] Curnow.

[10] Schmitt.

[11] McCready.

Sandifort, Obs. anat. patholog., Lugd. Batav., 1777, lib. iii. cap. iv. p. 73. *Cawley*, London Medical Journal, London, 1788, vol. ix. part iv. p. 286. *Salmade*, Recueil périodique de la soc. de méd. de Paris, 1797–1798, tome iii. p. 454. *Portal*, Cours d'anat. méd., Paris, 1803, tome v. p. 356. *Schmitt*, Zweifelhafte Schwanger, Wien, 1818. *Muhlbauer*, Neue Notizen (von Froriep), Weimer, 1822–1847. *Duponchel*, Soc. méd. d'Émulat., 1824, t. ix. p. 76. *Gendrin*, Histoire anat. des inflam., Paris, 1826, tome ii. p. 263. *Lecannan*, Journ. de pharm. et de chim., Décembre, 1827, p. 626. *Pemberton*, On Various Diseases of the Abdominal Viscera, Richmond, Va., 1830, chap. iv. p. 64. *Bécourt*, Recherches sur le Pancréas, Thèse, Strasbourg, 1830, § 5, p. 69. *Baillie*, The Morbid Anatomy of the Human Body, London, 1812, p. 115, and 1833, p. 222. *Bigsby*, Edinburgh Medical Journal, 1835, vol. xliv. p. 97. *Mondière*, Archiv. gén. de méd., II série, Paris, 1836, t. xii. p. 147. *Schupmann*, Hufeland's Journal, 1841, xcii. Bd. § iii. s. 41. *Engel*, Oesterreich med. Jahrbücher, 1841, xxiii. u. xxiv. Bd. *Wilson*, Med. Chirurg. Trans., London, 1842, vol. xxv. p. 42. *Devilliers*, Revue médicale, no. de Décembre, Paris, 1844, t. iii. p. 576. *Gross*, Pathological Anatomy, Phila., 1845, p. 689. *Gould*, Catalogue of the Anatomical Museum of Boston Society for Medical Improvement, Boston, 1847, §§ 575, 576, p. 173. *Schulze*, Journal für der Chemie, xxxix. p. 29. *Rokitansky*, Manual of Pathological Anatomy, Syd. Soc. London, 1849, vol. ii. § 2, p. 180; and Lehrbuch der Pathol. Anat., 1861, iii. s. 313, 369. *Clayton*, The Medical Times, London, 1849, vol. xx. p. 37. *Clark*, London Lancet, Aug. 15, 1851. *Dufrèsne*, Traité de l'affection calculeuse du foie et du pancréas, Paris, 1851, Article deuxième, p. 494. *Virchow*, Verhand. der med. physik. Gesellschaft zu Würzburg, 1852, II. Bd. s. 53 u. III. Bd. s. 368. *Virchow*, Die krankhaften Geschwülste, Berlin, 1863, I. Bd. s. 276. *Jones & Sieveking*, A Manual of Pathological Anatomy, Phila., 1854, p. 532. *Henry*, Journ. de chim. méd., IV. série, Paris, 1855, t. i. p. 273, et La France méd. et pharm., 3 année, Paris, 1856, No. vi. p. 42. *Jones*, Med.-Chirurg. Trans., London, 1855, p. 195. *Cruveilhier*, Traité d'anat. pathol. gén., Paris, 1856, t. iii. p. 365. *Ancelet*, Essai analytique sur l'anatomie pathologique du pancréas, Thèse, Paris, 1856, t. i. p. 26. *McCready*, New York Medical Journal, 1856, p. 78. *Hoppe*, Virchow's Archiv, 1857, xi. Bd. s. 96. *Parsons*, British Medical Journal, June 6, 1857, p. 475. *Recklinghausen*, Virchow's Archiv, Berlin, 1864, 30 Bd. s. 360. *Ancelet*, Études sur les maladies du pancreas, Paris, 1864. *Maier*, Archiv der Heilkunde, Leipsig, 1865, s. 168. *Jenner*, British Medical Journal, Jan. 16, 1869, p. 42. *Cornil et Ranvier*, D'Histologie pathologique, Paris, 1869–1873, tome ii. p. 974. *Pepper*, Centralblatt für die med. Wissensch., 1871, p. 156. *Delafield*, Handbook of Post-mortem Examinations and of Morbid Anatomy, New York, 1872, p. 203. *Janeway*, New York Medical Record, 1872, vol. vii. p. 357. *Curnow*, Trans. Pathol. Soc., London, 1873, vol. xxiv. p. 136. *Zenker*, Tagblatt der 47. Versam. deutsch. Natur. u. Aerzte in Breslau, 1874, s. 211. *Schmidt*, Annal de. Chem. u. Pharm., 1854, xcii. p. 33. *Wilkes & Moxon*, Lectures on Pathological Anatomy, Phila., 1875, p. 470. *Flint*, Text-book of Human Physiology, New York, 1876, p. 272. *Klebs*, Handbuch der Patholog. Anat., Berlin, 1876, I. Bd. s. 544 u. 547. *Carpenter*, Physiology, Phila., 1876. *Friedreich*, Ziemssen's Cyclopedia of Medicine, New York, 1878, vol. viii. pp. 615–618. *Cornil and Ranvier*, Manual of Pathological Histology, Translation, Phila., 1880, p. 581. *Foster*, Text-book of Physiology, American edition, Phila., 1880, p. 333. *Wm. Pepper*, Medical Ward Notes, 1880, Hospital of the University of Pennsylvania.

Omissions. *Galiati*, De morbus duobus observ., 1758, p. 26, or Comment. Bonon., t. iv. p. 34. *Rugg*, London Lancet, May 18, 1850. *Boch*, Lehrbuch der Pathol. Anat., Leipsig, 1847, § 7, s. 669.